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Cognitive impairments, emotion, stress, and language in schizophrenia

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Abstract

Language symptoms in schizophrenia are exacerbated by arousal of negative affect; the extent of this effect varies widely among patients. The present study assessed predictors of affective speech reactivity. Based on earlier research, it was expected that speech reactivity would be predicted by a combination of neurocognitive and emotional variables. We assessed patients ($n=50$) for baseline depression; neurocognitive functioning in the domains of sustained attention, immediate auditory memory, organizational sequencing, and conceptual sequencing ability; and clarity of speech communication in both stress and non-stress conditions. Twenty-three subject nominated significant others (SOs) also participated in the study, and were assessed for levels of expressed emotion (EE) as an index of relationship stressors. Patients, in turn, rated the subjective stressfulness of being in the presence of their SOs, from which the propensity to perceive interpersonal experiences as stressful was calculated by regressing out EE ratings. As predicted, baseline depression and sensitivity to interpersonal stressors were related to affective reactivity of speech, with stress sensitivity mediating the relationship between depression and speech reactivity. Contrary to expectations, baseline neurocognitive functioning was not related to speech reactivity. These findings are discussed in terms of their implications for understanding both schizophrenic language disturbance and stress vulnerability.

Keywords

Stress reactivity; affective reactivity; negative affect; speech disturbance; cognitive appraisal; schizophrenia

1. Introduction

Individuals with schizophrenia exhibit increased language disturbance when discussing emotionally negative topics, as compared to positive or neutral topics (Docherty and Hebert, 1997; Docherty et al., 1998; Cohen and Docherty, 2004; Burbridge et al., 2005). The clarity of *communicated meaning* has, in particular, been found to deteriorate under affectively negative conditions. Such deterioration has been termed affective reactivity of speech, because the induction of unpleasant affect by negative subject matter is the most plausible precipitating factor.

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Affective reactivity of speech is a differential phenomenon (Docherty, 1996). The speech of schizophrenic subjects as a group is more reactive than that of healthy controls. However, some schizophrenic individuals show only minimal reactivity, while in others it is quite marked. Affective reactivity of speech is associated with variables that have been implicated as process discriminators within the schizophrenic phenotype. These include greater familiarity of disorder (Docherty et al., 1998), higher than normal startle reactivity to acoustic stimuli (Docherty and Grillon, 1995), lower levels of deficit symptoms (Cohen and Docherty, 2004), and greater severity of the core positive syndrome of delusions and hallucinations (Docherty et al., 1996b). The factors underlying differential speech reactivity among schizophrenic individuals are not clearly understood.

A promising starting point from which to explore the underpinnings of speech reactivity is consideration of the cognitive causes of schizophrenic speech disorder, per se. Impairments in certain identified neurocognitive functions are believed to underlie a substantial proportion of the variance in schizophrenic communication disturbance. In a study that addressed these relationships, Docherty and colleagues (2000) found strong associations between indices of speech disturbance and the conceptually-related cognitive variables of sustained attention, immediate memory, and abstract-conceptual sequencing. Subjects who functioned poorly on these variables exhibited greater communication disturbance, overall, even after measures of global functioning and severity of illness were factored out. These findings were upheld and extended in a more recent study (Docherty et al., 2006). Given such findings, and given that affective reactivity of speech is predicated upon systematic variation in levels of communication disturbance – namely, increased communication failure under conditions of stress – it would appear reasonable to expect that baseline cognitive functioning might also be related to affective reactivity of speech. Preliminary evidence in support of an association between neurocognitive deficits and affective reactivity of speech comes from Burbridge and Barch (2002), who found that subjects with the poorest selective attention were the most likely to exhibit increased communication failure under stress. As these authors suggest, the negative affect associated with stress may overload already compromised neurocognitive functions in schizophrenic subjects, and thereby potentiate speech impairment.

Across a range of study designs and clinical populations, there is evidence to support a conceptually significant association between negative affect and stress reactivity (Bergdahl and Bergdahl, 2002; McLean, 2002; Hammen, 2005; Monroe and Harkness, 2005). Ilgen and Hutchison (2005), for example, found that individuals with a history of depression exhibited an increased sensitivity to stress when asked to solve an unsolvable problem. In a study using a schizophrenia sample, Myin-Germeys and colleagues (2000) found that subjects with relatively higher negative affect reacted more strongly to both actual and hypothetical life events. A central factor underlying such findings may be patients' core schematic belief structure, which, in both psychotic and non-psychotic depressives, is associated with negativistic appraisal processes biased towards the perception of threat (Fowler, 2000; Smith et al., 2006). Thus, one reason depressed subjects (schizophrenic or otherwise) may be relatively more stress reactive than non-depressed subjects is because they are predisposed to appraise situations in a negative and/or threatening manner. With respect to affective reactivity of speech, we suggest that cognitive appraisal functions as an important stress sensitivity variable, mediating the hypothesized relationship between depression and speech reactivity. In particular, the more schizophrenic subjects are depressed, the more they are likely to perceive situations as stressful; and the more they are likely to experience stress, the more they will manifest affective speech reactivity.

1.1 Present Study

The present study is an exploration of affective reactivity of speech, both as a unique behavioral phenomenon observed in schizophrenic patients, and as a putative effect of stress and stress sensitivity. The following relationships are hypothesized:

1. Affective speech reactivity will be associated with poorer functioning in each of the following neurocognitive variables: sustained attention, immediate auditory memory, organizational sequencing, and conceptual sequencing ability.
2. Affective reactivity will be positively associated with depression.
3. Affective reactivity will be positively associated with appraisal-based stress sensitivity.
4. The association between depression and affective reactivity will be mediated by appraisal-based stress sensitivity.

2. Method

2.1 Participants

2.1.1 Patients—Fifty outpatients from a larger research project (see Docherty et al., 2003; Cutting et al., 2006) participated in the study. Patients were selected if they met DSM-IV (American Psychiatric Association [APA], 1994) criteria for schizophrenia or schizoaffective disorder. Participants were excluded if there was evidence of current substance abuse, mental retardation, or history suggestive of organic complications (head injury, solvent abuse, etc.). The Schedule for Affective Disorders and Schizophrenia - Lifetime Version (SADS-L; Spitzer and Endicott, 1978), adapted for use with DSM-IV criteria, was used to make the diagnoses. All patients were receiving antipsychotic medications at the time of the study. Other selection criteria included a minimum global assessment of functioning rating (GAF; APA, 1994) over 30 (on a scale from 1 [worst functioning] to 100 [superior functioning]) and no psychiatric hospitalizations within the past three months. Demographic information for the patient sample is provided in Table 1.

2.1.2 Significant others—As the basis for obtaining stress sensitivity estimates (see Procedures), each participant was asked to designate the person whom they considered to have been the most influential in their lives over the past year, and who remained the most influential at the time of the interview. In selecting the significant other (SO), the perceived impact of this person was emphasized, rather than emotional closeness. In addition, it was required that subjects were currently interacting with their influential other at a minimum frequency of one time per week. Thirty-two of the subject-nominated SOs agreed to participate in the overall study. Of these, nine were excluded from the present analysis due to issues of missing data for the probands. The total effective subject pool for the SOs was, thus, twenty-three. Descriptive characteristics of the influential other sample are outlined in Table 1.

2.2 Procedures

2.2.1 Ratings of affective reactivity—Two 10-minute narrative speech samples were audiotaped, in which participants talked about affectively valenced memories. In the non-stress condition, subjects were asked to discuss “good” memories, whereas in the stressful condition, subjects were asked to discuss “bad” memories. Interviewers were present to provide the opening stimulus, as well as to keep subjects on task as needed and to elicit elaboration of condition-appropriate memories. The positive and negative narratives were collected on separate days to avoid carryover effects, and the order was counterbalanced. The narratives were transcribed and proofread to ensure accuracy.

Speech samples were analyzed using the Communication Disturbance Index (CDI; Docherty et al., 1996a). The CDI is designed to assess disturbances in communication of meaning, rather than thought disorder as such, or disorder of language structure. The CDI measures six conceptually distinct types of communication disturbances, namely, vague references, confused references, missing information references, ambiguous word meanings, wrong word references, and structural unclarities (for a discussion of these classifications, see Docherty et al., 1996a). Using the CDI, communication disturbances are assessed by first identifying discrete instances of failures in the transmission of meaning. The basic unit of analysis is the individual word or phrase, with the one exception being structural unclarities, in which agrammatical and/or incomprehensible combinations of words are observed. Instances of each type of communication failure are counted, following which CDI scores are determined as the frequency of communication failures per 100 words. Total CDI scores (aggregated across all six types of communication disturbances), were used in the present study and are an overall indication of communication disturbance. The CDI and its validation are described more fully in an earlier publication (Docherty et al., 1996a).

Affective reactivity of speech was assessed by regressing communication disturbance scores from the non-stressful condition onto scores from the stressful condition, and then deriving the standardized residuals. Residuals are preferable to difference scores as a measure of change in this context, because they represent differences in a proportionate manner. Due to the considerable variance that is found in patients' gross levels of referential disturbance, equivalent raw difference scores can have very different implications in terms of relative change.

2.2.2 Neurocognitive measures

2.2.2.1 Sustained attention: A degraded stimulus continuous performance task (CPT) was administered to each participant (Degraded Stimulus CPT, version 7.03, UCLA Clinical Research Center for the Study of Schizophrenia; based on the protocol described by Nuechterlein et al., 1983). A quasi-random series of digits is flashed on a computer screen, at the rate of one per second, for 8 minutes. Participants are instructed to watch the screen and to respond by pushing a button every time the target digit appears. It is believed that the use of degraded (blurry) stimuli imposes a relatively heavy information processing burden on subjects (see Nuechterlein et al., 1994). The test yields scores of hit rate, false alarm rate, sensitivity, and response bias. The sensitivity scores, which are derived from both hit rates and false alarm rates, were used as the measure of attention in this study.

2.2.2.2 Immediate auditory memory: Immediate auditory memory was measured by the digits forward portion of the digit span test from the Wechsler Adult Intelligence Scale-R (WAIS-R; Wechsler, 1981). This provides a measure of immediate auditory memory for simple sequences.

2.2.2.3 Organizational sequencing: Organizational sequencing ability was assessed by means of trail making test (Trails B; Reitan and Davidson, 1974). Trails B is a simple organizational task in which individuals are prompted to draw a series of lines connecting 26 letters and numbers in alternating order. In addition to basic sequencing ability, efficient performance on Trails B involves working memory, in that it requires the maintenance of a two-step task set in memory during task completion, and also an ongoing awareness of one's own latest responses (see Levitt et al., 1995). Performance is measured by time to correct completion of the task.

2.2.2.4 Abstract-conceptual sequencing: The capacity for abstract-conceptual sequencing was assessed using the conceptualization subtest of the Shipley Institute of Living Scale (SILS;

Shipley, 1940). This subtest consists of 20 sequences of numbers, letters, or words ordered on the basis of different underlying principles or rules. Subjects are required to demonstrate their understanding of the rule that underlies each sequence by generating missing items in the series. Blanks are provided at the end of each series, with one digit or letter per blank. Sequences are designed to become more difficult as individuals progress further in the test. Scores are computed as number of items correct, multiplied by 2.

2.2.3 Depression—Depression was assessed using the depression rating from the Brief Psychiatric Rating Scale (BPRS; Overall and Gorham, 1962).

2.2.4 Stress sensitivity—Stress sensitivity was operationalized using a novel methodology. The stressfulness of subject-nominated ‘most influential’ relationships, as assessed by objective ratings of expressed emotion (EE), was regressed out of subjects’ subjective ratings of the perceived stressfulness of the index relationship. It was posited that the resultant variable would represent subjects’ propensity to experience situations as stressful, independent of objective considerations, and would on that basis predict speech reactivity.

2.2.4.1 Expressed Emotion (EE) ratings: The Camberwell Family Interview (CFI; Brown and Rutter, 1966; Rutter and Brown, 1966; Vaughn and Leff, 1976) was used to measure EE, as this is the standard instrument for this purpose. Thus, each SO participant was administered the CFI in order to assess EE with respect to the target subject.

CFI ratings are based on two scales: criticism and emotional overinvolvement (EOI). Following CFI conventions, ratings of criticism were determined by the sum total of critical comments made about the patient during the CFI interview. SOs who made six or more critical comments were considered to be high on criticism (Brown et al., 1972). EOI is defined by the CFI as an exaggerated emotional response displayed as overanxiety, overprotectiveness, or overidentification with the patient; it is rated based on a scale from 0 to 5. An EOI rating of 4 or 5 is considered to be high. An SO scoring high on either criticism or emotional overinvolvement (or both) was classified as high-EE.

Details regarding the use of the CFI in this study are discussed at length in another paper (Cutting et al., 1996). Briefly, the CFI interviews were conducted by trained doctoral students who were blind to all other variables in the study. The audiotaped interviews were rated for EE by an individual trained to reliability at the University of Rochester. Similarly, a second trained rater independently rated 20% of the interviews, exhibiting very good agreement (Intraclass $r = .94$ for criticism; Intraclass $r = .90$ for EOI). The average number of days per week in which interactions took place between patients and their influential others was 4.9 ($s = 2.4$; range = 1–7).

2.2.4.2 Perceived Stress: Patients completed an item asking how “stressed” they felt when in the presence of their influential other. The item was rated using a 10-point Likert scale, anchored with the words “not at all stressed” at one end and “very stressed” at the other.

2.2.5 Timing of assessments—The measures used with the schizophrenia outpatients in the present study were administered over a two-day period. On day one, subjects were administered the SADS and BPRS, and provided a 10-minute speech sample in either the stress or non-stress condition. On day two, subjects undertook neurocognitive assessment, provided perceived stress ratings regarding their SOs, and provided the final 10-minute speech sample, under the opposite condition (stress or non-stress) to that provided earlier.

2.3 Analyses

Distributions of the CDI ratings were positively skewed, so we applied a logarithmic transformation before conducting comparisons. Correlations were used to assess the relationships between study variables and affective reactivity of speech. For the mediation hypothesis, Baron and Kenny's (1986) criteria were used: (1) the predictor (depression) must be significantly related to the potential mediator (stress sensitivity); (2) the predictor (depression) must be significantly related to the dependent variable (affective speech reactivity); (3) the potential mediator must be significantly related to the dependent variable; and (4) the relationship between the predictor and dependent variable must no longer be significant after controlling for the potential mediator.

3. Results

Table 1 contains descriptive information for the sample, expressed in terms of age, gender, race, education, global assessment of functioning (GAF) score, and Shipley IQ. None of these demographic variables were significantly related to affective reactivity of speech. Means and standard deviations for all study variables are displayed in Table 2; correlations of the predictor variables with the dependent variable, speech reactivity, are also shown in Table 2. Intercorrelations among the predictor variables are displayed in Table 3.

3.1 Neurocognitive functioning and affective reactivity of speech

Correlations were computed between the neurocognitive test scores and global affective reactivity on the CDI, as shown in Table 2. None of the neurocognitive variables were significantly associated with affective reactivity of speech.

3.2 Depression and affective reactivity of speech

As shown in Table 2, affective reactivity was significantly correlated with ratings of depression from the BPRS, $r=0.38$, $P<0.01$. Follow-up correlations were computed between depression and speech disturbance in both the stress and non-stress speech conditions. Depression was not significantly related to either of these variables ($r=0.09$ and $r=-0.23$, respectively), emphasizing the specificity of the relationship between depression and affectively reactive speech disturbance.

3.3 Expressed emotion ratings, perceived stress, stress sensitivity, and affective reactivity of speech

There was a wide range in numbers of critical comments made by SOs during the CFI, from 0 to 17, with a mean 4.75 ($s=5.37$). The mean EOI rating for the sample was 2.04 ($s=1.23$) on a scale of 0 to 5. Of the 23 SOs in this study, 9 were rated high in EE, based on their criticism and EOI ratings (39% of the sample), while 14 SOs (61% of the sample) were low in both criticism and EOI. For the 9 SOs rated high in EE, two SOs (9%) were high only in EOI, five (22%) were high only in criticism, and two (9%) were high in both EOI and criticism. As shown in Table 2, none of the EE variables were significantly associated with speech reactivity.

Subjects' perceived stress in the presence of their SO had a mean value of 2.96 on the 1–10 scale. Perceived stress was significantly associated with the theoretically-related predictor variables of SO EE, $r=0.41$, $P=0.05$, and depression ratings, $r=0.50$, $P=0.02$; and was related to the dependent variable, speech reactivity, at a marginal level of significance, $r=0.39$, $P=0.65$, indicated in Table 2. Stress sensitivity, which was calculated by regressing SO EE ratings out of the perceived stress variable, was correlated with speech reactivity, $r=0.49$, $P=0.02$.

3.4 Tests of mediation

A series of regression analyses were used to test the mediation model. The first regression found that depression significantly predicted stress sensitivity ($\beta = 0.452, t = 2.55, P=0.035$); the second regression showed that depression significantly predicted affective speech reactivity ($\beta = 0.375, t = 2.80, P<0.01$); and the third regression showed that stress sensitivity also significantly predicted speech reactivity ($\beta = 0.493, t = 2.53, P=0.02$). Thus, the basic conditions for mediation were met, based on Baron and Kenny's (1986) criteria. In a fourth regression analysis in which depression and stress sensitivity were entered to predict speech reactivity, stress sensitivity continued to predict speech reactivity ($\beta = 0.517, t = 2.314, P=0.032$) while the contribution of depression was no longer significant ($\beta = -0.053, t = -0.239, P=0.814$). These results are consistent with the mediation hypothesis.

4. Discussion

In accordance with expectations, depression ratings and stress sensitivity were related to affective reactivity of speech, and stress sensitivity was found to mediate the relationship between depression and speech reactivity. Contrary to expectations, baseline neurocognitive functioning was not predictive of speech reactivity using any of the four variables assessed..

Depression predicted speech reactivity, accounting for 14% of the total variance. This finding is consistent with a large body of research substantiating complex longitudinal relationships between stress, emotions, and psychosis. During acute psychotic episodes, depressive symptoms have been found to increase and decrease with the onset and offset of psychotic symptoms (Green et al., 1990; Koreen et al., 1993; der Heiden et al., 2005). This relationship suggests that depression can occur as an effect of psychotic exacerbation. There is also evidence that depressed mood can independently contribute to the development of psychosis (Freeman and Garety, 2003; Krabbendam et al., 2005; Smith et al., 2006). Based on such evidence, Garety and colleagues (2003) argue that negative emotion has a normal and direct role in the development and maintenance of psychotic symptoms. They posit that emotional processes involved in psychotic decompensation operate akin to those of non-psychotic depressives, for whom low mood is associated with distorted appraisal and thought processes. The positive relationship between depression and stress-induced speech reactivity is consistent with this conceptualization. Stress reactivity, as behaviorally operationalized in the present study (increased communication disturbance under a negatively valenced speech condition), may represent the outward manifestation of depressive appraisal processes in which situations are more likely to be perceived as stressful.

The study's findings regarding stress sensitivity allow us to refine this interpretation. By removing the effects of differences in actual EE levels from the perceived relationship stress variable, we created the residualized perceived stress variable to capture individual differences in the propensity to experience stress, independent of objectively assessed stressors. Once shared variance with the objectively-assessed stressfulness of a significant relationship was removed from perceived stress ratings (note: perceived relationship stress and SO EE were significantly positively correlated), we expected that the residual variance would capture individuals' appraisal tendencies. This expectation was supported by the fact that perceived stress ratings were more strongly associated with speech reactivity after controlling for observed levels of EE. Consistent with our hypotheses, moreover, the residualized stress sensitivity variable was significantly related to affective reactivity of speech, and was also found to mediate the relationship between depression and speech reactivity. In regression analysis, when both stress sensitivity and depression were entered as predictors of speech reactivity, the effect of stress sensitivity on speech reactivity remained significant, whereas the contribution of depression ratings was no longer significant. Because stress sensitivity, as operationalized in the present study, is an implicit function of idiosyncratic appraisal processes,

we believe the present results support a primary role of cognitive appraisal processes in schizophrenic stress reactivity.

The cognitive appraisal interpretation of our findings regarding speech reactivity offers an interesting parallel to the speculations of Fowler (2000), who concluded that negative schematic beliefs increase the risk for distressing auditory hallucinations and persecutory delusions independent of depressed mood. In a recent study, moreover, it was found that high stress reactivity increased the risk for psychotic symptoms following life events (Docherty et al., 2007). The latter study measured stress reactivity through self-report, whereas in the present study stress reactivity is imputed to the phenomenon of affective reactivity of speech, a uniquely behavioral measure. Are both operationalizations capturing the effects of biased cognitive appraisal? While the data do not allow for a conclusive answer to this questions, the cognitive appraisal interpretation is theoretically grounded, integrative, and parsimonious. Negative core schemata and cognitive appraisal biases may have broad implications for individuals with schizophrenia-spectrum disorders, increasing the generalized risk for the experience of stress, while also influencing the risk for positive symptoms, their specific content, and their associated distress. These considerations are offered tentatively. While the present results are consistent with cognitive-based conceptualizations of the effects of stress and depression, and particularly with the cognitive model of psychosis proposed by Garety et al. (2001), future research is required to replicate and extend the present preliminary findings.

The null findings regarding neurocognitive functioning can be contrasted with earlier research which has found strong associations between neurocognitive functioning and gross levels of communication disturbance, under both stress and non-stress conditions (e.g., Docherty et al., 2006). In this regard, affective reactivity of speech, understood as a change phenomenon defined by *increased* communication failure under stress, appears to be quite different from communication failure, which does not incorporate the idea of change or stress reactivity. Results in the present study are consistent with the interpretation that differences among patients in affective reactivity are based largely on emotional-psychological phenomena. Thus, relatively poorer functioning in the neurocognitive variables assessed does not appear to confer increased liability to increased communication failure under negative emotional conditions. Notably, the null result regarding the sustained attention variable may appear to be at odds with the findings of Burbridge and Barch (2002), who found that selective attention significantly predicted speech reactivity. However, it should be emphasized that these are conceptually distinct attention variables. Whereas sustained attention measured through the degraded-stimulus CPT is primarily an assessment of vigilance, selective attention measured through errors and response times on the color Stroop task (Burbridge and Barch) assesses the ability to quickly select the appropriate dimension of a stimulus and to inhibit the irrelevant dimension (Henik et al., 2002).

Given existing research and theory about the neurocognitive underpinnings of speech communication (for a concise review, see Docherty et al., 2000), it would appear implausible to conclude that neurocognitive variables such as sustained attention, memory, sequencing ability, and abstract reasoning ability are not critically involved in affectively reactive speech disturbance, albeit at a secondary level. Following the work of Burbridge et al. (2005), we suggest that rather than making an independent contribution to speech reactivity, neurocognitive functioning plays a mediatory role. In particular, we suggest that as speech-critical neurocognitive functions such as attention and memory are *temporarily worsened* by the individual's stress response (which may in turn be moderated by individual-level factors such as depression and threat appraisal), gross levels of speech disturbance are increased. Because this kind of neurocognitive mediation would be limited to the proximal, 'in-the-moment' stress response, baseline measures of neurocognitive functioning may not predict

speech reactivity to the extent that they have been shown to predict non-reactive impaired communication.

The main limitation to this study is its cross-sectional design, which does not allow for the derivation of strong inferences regarding causal relationships. Another limitation is the small sample size, which limited power to detect significant associations, particularly for analyses that incorporated SO ratings ($n=23$). The SO sample was heterogeneous in terms of relationship categories (parent, spouse, friend, etc.), and the variability introduced by that heterogeneity may have weakened results in terms of the derived stress sensitivity variable. Although efforts were made to ensure that patients chose their most influential person for inclusion in the study, regardless of whether the influence was positive or negative, the very nature of selecting an associate whom they consider to be the “most influential” in their lives may have biased the selection process in the direction of relationships that subjects experienced as positive. Finally, it should be noted that because all patients participating in this study were receiving pharmacotherapy at the time of the assessment, their stress reactivity may have been suppressed to some extent, thereby attenuating speech reactivity. Moreover, assuming that medication status might exert varying levels of effect on key study variables (e.g., speech reactivity; perceived stress; depression; etc.), it is possible that the observed associations would differ in both absolute and relative magnitude using a non-medicated sample.

This study offers new information about the role of negative affect in affective reactivity of speech, suggesting that baseline levels of depression and appraisal-based stress sensitivity can be important determinants of vulnerability to exacerbation of disorganization symptoms under conditions of stress. The failure to find significant associations between baseline neurocognitive functioning and speech reactivity suggests limits on the extent to which communication disturbance can be understood independently of psychological functioning.

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Table 1

Descriptive Information for Sample

	Patients	Significant others
No. of Subjects (<i>n</i>)	50	23
Mean Age (in years)	36.36 ± 9.77	50.8 ± 13.9
Mean Years of Education	12.24 ± 1.47	13.22 ± 2.26
Mean GAF score	50.68 ± 11.94	77.13 ± 8.13
Mean Total Number of Hospitalizations	8.11 ± 8.24	N/A
Mean # of Months Since Hospitalization	45.08 ± 53.25	N/A
Mean Age at First Psychiatric Treatment	20.16 ± 6.29	N/A
Mean Age at First Hospitalization	22.58 ± 6.08	N/A
% Female	42	73.9
% Caucasian	71.4	78.3
% African American	28.6	21.7
% of Significant Others: Parents	N/A	52
% of Significant Others: Siblings	N/A	22
% of Significant Others: Spouses	N/A	13
% of Significant Others: Friends	N/A	9
% of Significant Others: Romantic Partners	N/A	4

Table 2
Means and Standard Deviations of Predictor Variables, and Correlations with Speech Reactivity

Construct	Measure	Variable	n	M (± SD)	Correlation with affective reactivity of speech
Neurocognitive functioning					
Sustained attention	Degraded stimulus CPT	Sensitivity (A')	50	0.80 (± 0.13)	-0.09
Immediate memory	Digit span – digits forward	Number correct	50	6.68 (± 1.97)	0.04
Organization/sequencing	Trail making test B	Time to completion	50	134 (± 67)	0.17
Conceptual sequencing	Sequence completion – Shipley	Total correct	50	17.96 (± 9.62)	-0.13
Depression	BPRS depression rating	Likert rating, 1 – 7 (1 = “not present”)	50	2.40 (± 1.29)	0.38 **
Relationship stress					
Criticism	CFI: EE criticism	Number of Critical Comments	23	4.75 (± 5.37)	-0.25
Emotional overinvolvement	CFI: EE emotional overinvolvement	Likert rating, 0 – 5 (0 = “no emotional overinvolvement”)	23	2.04 (± 1.23)	0.06
Expressed emotion	CFI: Total EE rating (high/low)	Rated “high” if 6 critical comments made and/or an EOI rating of 4 or above	23	n/a	-0.06
Perceived stress	Question: “How stressed do you feel in the presence of your significant other?”	Likert rating, 1–10 (1 = “not at all stressed”)	23	2.96 (± 2.33)	0.39
Stress sensitivity	Calculated by regressing EE ratings out of perceived stress ratings	n/a	23	n/a	0.49 *

* Correlation significant at the 0.05 level (2-tailed)

** Correlation significant at the 0.01 level (2-tailed)

Table 3

Correlations Among Predictor Variables

	1	2	3	4	5	6	7	8	9	10
1. Sustained attention	1									
2. Immediate memory	-.040	1								
3. Organization/sequencing	-.40**	-.027	1							
4. Conceptual sequencing	0.22	0.31*	-.056**	1						
5. Depression	-.002	-.014	0.05	-.021	1					
6. EE criticism	-.48*	-.012	-.002	-.008	-.005	1				
7. EE EOI [†]	-.005	0.02	-.035	0.40	0.13	0.20	1			
8. EE - total	-.036	-.019	-.008	-.003	0.24	0.83**	0.57**	1		
9. Perceived stress	0.24	-.003	-.014	-.028	0.50*	0.10	0.26	0.41*	1	
10. Stress sensitivity	0.43	0.04	-.012	-.029	0.45*	-.027	0.03	0.000	0.92**	1

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

[†] EOI = Emotional overinvolvement